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RISKS ASSOCIATED WITH PATHOGENS IN COMPOSTED BIOSOLIDS

A Discussion Paper Prepared for the Water Authority of Western Australia

by

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SUMMARY

Information available from published epidemiological studies, laboratory studies and field studies was surveyed and it was concluded that there was not sufficient evidence to suggest that the composting process completely removed the risk associated with the unrestricted marketing of biosolids to home gardens. A risk assessment was therefore carried out to develop criteria for acceptable concentrations of pathogens in composted biosolids products. A number of principles were developed for the risk assessment. One of the principles was that the most at risk individuals should be protected and it was decided that these would be young children playing in home gardens. Another principle was that microbial risk assessments should be based on risks of disease, rather than risks of death or risks of infection. The principle adopted for deciding acceptable risk was that the risk of disease transmission through the re-use of composted biosolids products should be less than background transmission rates for that disease from other sources.

The above risk assessment approach was used to develop suggested limits for *Salmonella* in composted biosolids products. The suggested limit is less than 1 *Salmonella* in 50 g of biosolids product. A recommendation is that guidelines should also require a maturation period for composted biosolids. Further research on the regrowth potential of *Salmonella* in composted biosolids products is recommended.

Although there is a high potential risk associated with *Giardia* and enteric viruses in composted biosolids, it is recommended that guidelines should not require the monitoring of composted biosolids products for *Giardia* or enteric viruses until methods are further developed.

It is also recommended that an epidemiological study of the effect of the unrestricted marketing of composted biosolids on the spread on enteric disease should be carried out.

LIST OF CONTENTS

LIST OF CONTENTS (Continued)

LIST OF TABLES

1. INTRODUCTION

'Biosolids' is a term which was introduced by the US Water Pollution Control Federation to replace the generic term sludge (Smith, 1995). The NSW EPA has used this term in a more restricted sense to refer only to sludge derived from wastewater treatment (EPA NSW, 1994). As there is a difference in the scope of the sludge material covered by these two definitions, it was decided that in this paper the term biosolids will refer to material previously known as sewage sludge or wastewater sludge.

This paper is a discussion of the risks associated with pathogens in biosolids disposal and re-use, but is limited in its scope. The first limitation is that this paper concentrates on human pathogens and does not deal with risks from pathogens of animals and plants. Secondly, this paper is primarily concerned with one disposal route for biosolids, which is the unrestricted marketing of biosolids. This paper also focuses on the composting process as a means of producing biosolids suitable for unrestricted marketing.

2. POTENTIAL RISKS FROM PATHOGENS IN BIOSOLIDS

There is still a great deal of uncertainty about risks associated with pathogens in biosolids products. Biosolids material has the potential to contain human and animal pathogens as it contains human faecal material. It has been demonstrated that pathogens can be present in a variety of biosolids products (Gibbs *et al.*, 1995). However, what level of risk this presents is still uncertain.

There is also a climate in Australia where the water industry has to face the new and complex issues of 'liability, precautionary principles, due diligence and what constitutes reasonable care' (Chapman, 1995). Liability of individual directors may increase due to loss of immunity when water authorities are corporatised or privatised. This means insurance cover must be increased or risks reduced.

An outbreak of waterborne cryptosporidiosis in Milwaukee in 1993 illustrates the problems which can result from an outbreak of disease for which the water provider is held responsible. This outbreak resulted in over 400 000 cases of cryptosporidiosis (MacKenzie *et al.*, 1994) with 50 to 100 deaths and has accumulated approximately $25 000 000 in lawsuits (Chapman, 1995).

Along with illustrating the responsibility that the water industry has for the products which they supply, the Milwaukee outbreak highlights the importance of adequate monitoring for appropriate pathogens or indicators. Traditionally the microbiological quality of drinking water has been assessed using the coliform and faecal coliform tests, and standards based on these are in place throughout the world. In Milwaukee these proved to be inadequate. In the case of biosolids products, monitoring is not routinely carried out. There is not a traditional framework for assessing the risks associated with pathogens in biosolids and one needs to be developed.

3. MANAGEMENT OF RISKS FROM PATHOGENS IN BIOSOLIDS

As outlined in Section 2 there is still a great deal of uncertainty about the risks to human health posed by the disposal and re-use of biosolids. At present there is not agreement about how the potential risks of pathogens in biosolids should be managed.

In most existing guidelines for biosolids management, management procedures are based on classification of biosolids in terms of the treatment the biosolids products have undergone, and then what disposal route is permissible.

The proposal of this paper is that when it comes to risks of pathogens in biosolids, it may be useful to take a slightly different position and devise biosolids guidelines on the basis of what the level of exposure to the biosolids is going to be. Different disposal routes could be classified on the basis of the exposure level they present, and then for each exposure level and disposal route a combination of treatments and/or monitoring requirements specified which would make that particular disposal route acceptable. This would probably result in similar management practices to those presently in existence, but provide a stronger theoretical framework. Although this paper only deals with human pathogens, risks from animal and plant pathogens could be included in this framework.

On the basis of categorising the human exposure levels to biosolids products the following three categories are therefore proposed.

The **first category** is **very limited exposure**. Included in this category could be disposal practices such as landfilling or limited access minesite rehabilitation.

The **second category** is **limited exposure**. This includes the use of biosolids in various disposal practices where public access is possible, to varying degrees, but limited. This category could, and probably should, be divided into further sub categories. Included in this category could be disposal routes such as tree farming, market gardening, landscaping of public spaces etc.

The **third category** is **unrestricted exposure**. This category is for the unrestricted marketing of biosolids where the possibility of public exposure is high.

It is suggested that a management framework and standards should be developed for each exposure category on the basis of an informed analysis of the risks. This should be based on a combination of epidemiological information and risk analysis.

This paper will attempt to provide such an analysis for the **third category**, that of **unrestricted exposure**. The disposal practice which results in unrestricted exposure is the unrestricted marketing of biosolids products.

3.1 MANAGEMENT OF RISKS ASSOCIATED WITH UNRESTRICTED MARKETING OF BIOSOLIDS

The philosophy endorsed in most biosolids guidelines is that for biosolids to be available in a unrestricted manner to the public they should have undergone significant treatment for stabilisation and reduction in pathogen concentrations. The most detailed information and guidelines for the unrestricted marketing of biosolids have been produced by the United States Environmental Protection Agency (US EPA). In a support document for earlier federal sludge regulations, processes to further reduce pathogens (PFRPs) and process requirements for the PFRPs were outlined (US EPA, 1989a). Biosolids which were available for unrestricted marketing had to have undergone a PFRP. In more recent sludge guidelines, monitoring requirements have been introduced for biosolids which are available for unrestricted marketing, and these are described in another support document (US EPA, 1992). The more recent document has introduced requirements to reduce pathogen densities below certain concentrations. These concentrations were selected on the basis of the limits of detection methodologies (US EPA, 1992).

The major critique of this approach, and one of the reasons for preparing this paper, is that if monitoring requirements are introduced in biosolids guidelines, then the limits should take into account an analysis of risks posed by the pathogens present in biosolids. For this reason, this paper introduces a risk assessment approach for devising limits. The methodological limitations also need to be taken into account when setting standards. However, if requirements are only based on method limitations then requirements may be too severe if methods are very advanced, or not adequate if methods are not well developed. This risk assessment approach is introduced in Section 4.2 below.

A number of processes may provide pathogen reduction to a level which makes biosolids possibly suitable for unrestricted marketing, as outlined by the US EPA (1992). Two such processes outlined by the US EPA (1992) are thermal treatment and high pH-high temperature processes. The full range of processes is not discussed further in this paper but it is proposed that monitoring requirements developed for composted biosolids could be transferable to other processes, as they are based on the principle of acceptable risk. The focus of this paper is on the composting process and the rest of this paper is devoted to addressing the risks associated with pathogens in composted biosolids.

4. COMPOSTED BIOSOLIDS

There are two basic approaches which can be used for assessing the risks associated with biosolids products. The first is an epidemiological approach and the second a risk assessment approach. These are discussed below.

4.1 EPIDEMIOLOGY

Epidemiological information in all areas of biosolids disposal is scarce. The main reason for this is that costs involved in epidemiological studies are extremely high. Costs involved in prospective studies described by Jakubowski (1986), which were conducted in the late 1970s and early 1980s, ranged from US$178 000 to US$2 000 000.

The epidemiological studies which have been conducted, or reported cases where disease appears to have been spread by biosolids, are primarily associated with the use of biosolids on agricultural land. On the basis of the US epidemiological studies described by Jakubowski (1986), risks appear to be low. However there do appear to be cases where disease has been associated with biosolids disposal. Block (1986) described a case where four men spreading biosolids on farmland became infected with hepatitis A. An outbreak of 98 human cases of salmonellosis appeared to have been caused by drinking unpasteurised milk from a farm in Scotland (Pike, 1986). Biosolids containing effluent from a chicken factory had been sprayed on grassland and cattle re-introduced shortly afterwards. Another milk born epidemic in Czechoslovakia (Raska *et. al.*, 1966) appeared to have been caused by biosolids spread on land.

These small number of studies have shown that biosolids used on agricultural land can cause disease. However, if guidelines are followed which include biosolids treatment or restrictions on the use of biosolids amended areas and public access, then the health risks appear to be low.

This view has been strongly expressed by two British writers. Alderslade (1981) felt that surveillance of the incidence of disease, and investigation of outbreaks were effective in protecting public health. Similarly (Pike, 1981) felt that operational guidelines were sufficient to protect the public from the spread of disease from biosolids reuse. This view is supported by EC (1986) and UK (DOE, 1989) sludge guidelines.

There appears to have been no studies of the health effects of the unrestricted marketing of biosolids for home use. One study was conducted which examined the health of biosolids compost workers (Clark *et. al.*, 1984). Workers involved in composting showed evidence of an immune response to antigens which was higher then groups not involved with compost activities. More symptoms of burning eyes and skin irritation were also reported among compost workers. However, these may have been associated with high dust levels rather than biosolids.

There appears to have been no epidemiological studies which have investigated the health effects of unrestricted public exposure to composted biosolids. This would be a valuable area of investigation.

4.2 POTENTIAL RISK

As there is no epidemiological information which can be used to evaluate the risks associated with the unrestricted marketing of composted biosolids, it is useful to examine the literature concerning the effect of composting on pathogen concentrations in biosolids.

A number of laboratory studies and a small number of larger scale studies have investigated the effect of composting on pathogen concentrations in biosolids. Most of these studies have focused on salmonellae. No published information appears to be available on the effect of composting on *Giardia* cyst concentrations, although this appeared to be the pathogen which presented the most potential risk in dewatered biosolids (Gibbs *et. al.*, 1995).

Until recently it was assumed that biosolids which had met US EPA composting criteria (US EPA, 1989a) would be free of pathogens. Pederson (1981) concluded that composting was a satisfactory means of achieving biosolids disinfection, provided that specified temperatures were maintained throughout the compost pile. However, evidence provided by Yanko (1988) and Skavanis and Yanko (1994) suggests that this may not be the case. In a study by Yanko (1988) biosolids based compost products from 26 different composting facilities were sampled over the course of one year. *Salmonella* spp., toxigenic *E.coli* and *Yersinia enterocolica* were detected. Of these, salmonellae were the most commonly detected (occurring in approximately 20% of samples). Similarly, more recently it

was reported that salmonellae were detected in 25% of composted biosolids products (Skavanis and Yanko, 1994). The composting facilities were meant to be operating according to US PFRP guidelines.

These studies suggest that achievement of operational criteria may not guarantee the safety of composted biosolids products. One of the major problems may be that time and temperature requirements are difficult to achieve uniformly through entire batches of composted biosolids. Another possibility may be that composting does not ensure the complete removal of salmonellae, which are present but undetected using current methodology.

In a recent study of composted biosolids in Western Australia, although composting process requirements were achieved in some sections of a forced aeration static pile composting system, in situ temperature monitoring showed that this was not achieved for complete batches of biosolids (Mort, personal communication). In this study it was found that nearly all samples of final composted biosolids were positive for *Salmonella* (Mort, personal communication). In small scale batch studies using the same mixture it was found that *Salmonella* grew in composting mixtures. In this small scale system *Giardia* cyst concentrations decreased but *Giardia* was not completely removed by the composting process. Cyst concentrations were not reduced below 1000 cysts per gram of composted biosolids (Mort, personal communication).

The studies described above suggest that process requirements may not ensure the complete removal of pathogens during composting processes, but this should be investigated further. It appears that monitoring may also be necessary to ensure the safety of biosolids products. For this reason the risk assessment approach is introduced below and then applied to developing monitoring criteria for pathogens in composted biosolids.

4.3 RISK ASSESSMENT APPROACH

Risk assessment has been defined in a number of ways but the definition which has been applied in risk assessments associated with biosolids is that provided by the US National Research Council (NRC, 1983). Their definition is that risk assessment is:

> **"the characterization of the potential adverse health effects of human exposures to environmental hazards".**

This risk approach was defined by the US National Research Council (NRC, 1983) as consisting of four components. These were:

i. **Hazard identification**:
The process of determining whether exposure to an agent can cause an increase in the incidence of a health condition. As discussed in a US EPA document describing the risk assessment methodology for sludge (US EPA, 1989b), this should also include reference to the nature and severity of the effect as well as the incidence.

ii. **Dose-response assessment**:
The process of characterising the relations between the dose of an agent and the incidence of the adverse health effect.

iii. **Exposure assessment**:
The process of measuring or estimating the intensity, frequency and duration of exposures to an agent currently present, or estimating hypothetical exposures that may arise.

iv. **Risk characterization**:
Performed by combining the exposure and dose-response assessments to estimate the likelihood of an effect.

This approach was used by the US EPA as the model for developing guidelines for sludge disposal (US EPA, 1989b).

Although the risk assessment approach has been defined for chemicals in biosolids (US EPA, 1989b), it has not been well developed for microbials. For this reason, before a risk assessment could be carried out, some principles needed to be developed. These are introduced below.

4.3.1 Microbial Risk Versus Chemical Risk

Many of the basic assumptions used for chemical risk assessments may not be directly transferable to assessing microbial risk.

As outlined in the reference document to the development of US EPA sludge guidelines (US EPA, 1989b), determining acceptable concentrations for non carcinogenic chemicals involves determining the critical systemic effect, which is the adverse effect occurring at the lowest dose. The reference dose (RfD) is the

daily exposure that is likely to be without appreciable risk of deleterious effects during a lifetime.

Similarly in draft Australian Drinking Water Guidelines (NHMRC and ARMCANZ, 1994) the guideline values for pesticides and organic and inorganic chemicals are the concentrations that, based on present knowledge, do not result in any significant risk to the health of the consumer over a lifetime of consumption (the no observable effect level, NOEL).

There are a number of ways in which this approach is probably not appropriate for microbials and these are described below.

4.3.1.1 Lifetime Exposure Models

The cumulative lifetime exposure model used for chemicals is probably not appropriate for microbials. Single hit exposure to microbials can result in an adverse health effect and effects are generally not cumulative. Previous exposure may cause immune suppression which could increase the likelihood of infection, or conversely previous exposure may result in the development of immunity which will reduce the effect of repeated exposures. However, it was not considered possible to incorporate these effects into a risk assessment model, so a single hit exposure model is recommended.

4.3.1.2 Population Versus Individual Risk

Chemical exposure models do not take into account variation in human host, response which is an important factor with microbial exposure. With microbials the large variation in susceptibility will significantly affect risk. For this reason, expressing risk in terms of general population risks is probably not appropriate for microbials. Risks need to be expressed for individuals within sub-groups of the population.

As outlined in a discussion paper on risk assessment produced by the Western Australian EPA (1990), the principle that no individual should have to bear an unusually high risk can be expressed by individual risk criteria. Secondly the principle that no community should have to bear an unfairly high risk can be expressed by 'community', 'societal' or 'population' risk criteria. Although it may also be useful to investigate population risks, this paper focuses on

individual risks. This is because, in the case of microbials in biosolids, individual risk criteria will need to be more stringent than population risk criteria.

Therefore the first suggested principle is that:

> **Principle 1.**
> **Risk assessments for microbials in biosolids should be based on risks to the most at risk individuals.**

Principle 1 should be applied in two areas of the risk assessment. Firstly exposure levels should be based on most exposed individuals. Secondly dose-response data should be based on most susceptible individuals.

This principle of protecting susceptible or vulnerable individuals has been applied in the area of industrial exposure. For assessing the safety of location of a proposed development of a potentially hazardous nature, the criteria shown in Table 1 were suggested (Western Australian EPA, 1990). Table 1 was developed on the basis of variations in people's vulnerability to the risk and their ability to take evasive action. Although Table 1 is not directly applicable to risk criteria for disease rather than death, it introduces the principle of protecting the most at risk individuals.

Table 1. Suggested Individual Fatality Risk Criteria for Acceptable Risk From Industry on Various Land Uses (Western Australian EPA, 1990)

Land Use	Suggested Criterion (risk in a million/year)
Hospitals, schools, child-care facilities, old age housing	0.5
Residential, hotels, motels, tourist resorts	1
Commercial developments including retail centres, offices and entertainment centres	5
Sporting complexes and active open spaces	10
Other industrial	50

The approach of calculating risk for most at risk individuals has been questioned by various sources because it can lead to an unrealistic amplification of risk. The US EPA (1989b) outlined a drawback of this approach and this was that the compounding of worst-case assumptions may lead to improbable results. They suggested that the key to the effective use of this methodology is careful and systematic examination of the effects of varying each of the input parameters, using estimates of central tendency and upper-limit values to gain an appreciation for the variability of the result.

Similarly Stevens *et al.* (1995) suggested that Monte Carlo simulation should be used to allow consideration of the variability of input parameters used in the risk assessment. With this approach, rather than assigning worst case or typical values, a probability distribution is assigned to each parameter.

In the case of microbials the representation of uncertainty in estimates would be valuable. However, it is still maintained that this should be carried out for individuals within different sub populations, rather than representing the whole population by one analysis. For the most at risk individuals within the sub population uncertainty of estimates could be expressed, and Monte Carlo type analysis for representing uncertainty may be appropriate.

4.3.1.3 Mortality and Morbidity

Most chemical risk assessments of health effects are based on the risk of death (mortality). However, it is suggested that risk assessments based on adverse health effects (morbidity) are more appropriate to microbial risk assessment. The risks of death from microbial exposure may be low for most infectious agents, but the adverse affects caused by disease still unacceptable to the community.

An added dimension with microbials is that exposure to a microbial agent may result in infection but not demonstrable illness. This may then impact on the general health of the community by providing a reservoir of disease for further infection. As this is very difficult to assess it is suggested that risks should be based on disease rather than infection.

Therefore the second principle which is suggested is that:

Principle 2.
Microbial risk assessments should be based on risks of demonstrable disease, rather than risks of death, or infection.

As there is not such an established history of assessing risk of disease rather than death, there are not well established practices, particularly with regard to risk management decisions. This will be discussed further in Section 4.4.2 below.

4.4 APPLICATION OF RISK ASSESSMENT APPROACH TO SETTING CRITERIA

When the four steps of risk assessment outlined in Section 4.3 above are carried out, a hazard is defined, and the risks then quantified through dose-response and exposure assessment. To use the risk assessment approach for developing monitoring criteria the same process needs to be carried out but acceptable risks also need to be defined. Following hazard identification, the next step in using risk assessment to develop criteria is to define an allowable or acceptable exposure. The dose-response and exposure level then need to be combined with the acceptable risk to set standards. The steps involved in developing criteria for microbials in biosolids therefore are:

1. Hazard Identification
2. Development of Criteria for Acceptable Risk
3. Dose-Response Assessment
4. Exposure Assessment
5. Calculation of Limits.

4.4.1 Hazard Identification

A qualitative risk assessment was carried out previously in which the relative health risks represented by different groups of pathogens were grouped on the basis of the number of reported cases (in Western Australia), the excreted load, the persistence, and infectious dose (Gibbs and Ho, 1993). On the basis of this assessment the high risk group of pathogens consisted of enteric viruses, and the second highest risk group of *Salmonella*, *Giardia* and *Trichuris*. As the people infected with *Trichuris* were mainly recent immigrants and travellers, it appeared that the number of excreting individuals in the resident population

were low. Further studies were therefore conducted on the pathogens considered to present the most risk, the enteric viruses, *Salmonella* and *Giardia*. These pathogens were detected in anaerobically digested and dewatered biosolids with average concentrations of 5.3, 0.4 and 920/g wet weight respectively. On the basis of infectious dose information provided by Shuval *et. al.* (1986) risks of infection from ingesting 0.1 g of biosolids were calculated to be less than 1% for viruses and *Salmonella*, and greater than 1% for *Giardia*.

In the absence of any information about the effect of composting on viruses and *Giardia*, and on the basis that *Salmonella* appears to have survived and regrown in some composting processes, stored biosolids and biosolids applied to land (Gibbs *et. al.*, 1995), it is likely that these pathogens will also present the most risk in composted biosolids. It is therefore suggested that these should be included in considerations of setting criteria for monitoring composted biosolids.

4.4.2 Acceptable Risk

The meaning of acceptable risk can be expressed in various ways. Commonly it is expressed as the question: 'how safe is safe enough'? How safe is safe enough has not been defined previously for the area of microbial risk and is still a contentious issue for most areas of risk.

Acceptable risks have generally been defined in terms of risk of death and this approach is described below. However, the principle recommended above is that with microbial risk, acceptable risk as it relates to disease should be used as an alternative approach, so this is also discussed below.

The approach taken by a Royal Society Study Group (1983) to defining the concept of acceptable risk was to divide the risks to the individual into three broad zones. In category one were risks higher than a certain level which were unacceptable whatever the benefits. In category two were risks that were between upper and lower limits and for which it was necessary to consider the interrelationship of level of risk, detriment, costs and benefits. In the third category were risks of a frequency so low that the manager or regulator of risk could reasonably regard them as negligible in their overall impact on society, even though the consequences to the rare individual might be serious.

The report also discusses some possible quantitative guidelines. These were:

1. The Upper Limit of Risk to an Individual.
   A continuing risk of death of 1 in 100 would be described as unacceptable in essentially all circumstances.

2. Risks Between Upper and Lower Limits.
   A risk of 1 in 1000 was not considered to be totally unacceptable in all circumstances, as long as the individual understood the risk, and the benefits, and that everything reasonable had been done to reduce it. However, risks in the borderline of unacceptability would require the most stringent process of justification before they could be accepted, particularly if they did not arise from the voluntary choice of the individuals affected.

3. The Negligible Level of Risk to an Individual.
   The point at which an imposed risk could be legitimately treated as trivial by a decision maker could be judged as the point at which individuals who were aware of the risks they run would not commit significant resources of their own to reduce them. Individuals would have widespread views but there is a commonly held view that few would take action at the annual level of 10^{-6}.

On the basis of these figures, risk management would be used to compare risks, detriments, costs and benefits, if the annual risks of death to the individuals were between 10^{-3} and 10^{-6}.

As this approach refers to risk of death, it is not directly transferable to defining acceptable risks of disease. However, it does introduce the principle of 'de minimis' risk, or negligible risk. The 'de minimis' risk approach comes from the legal principle that the law does not concern itself with trifles. With risk this means that there is a threshold of concern below which we would be indifferent to changes in the level of risk (Fiksel, 1987). It is an acceptance that zero risk is neither achievable or desirable.

There have been a variety of suggested means of setting acceptable risks on the basis of 'de minimis' risk. These include setting levels at natural background levels, such as for radiation, and alternatively basing acceptable risks on the risk levels of various hazards that are commonly encountered in daily life (Menkes and Fray, 1987). The assumption is that these levels of risk are acceptable to the population.

An acceptable limit for risk of infection from microbials was recently set for the first time when a 'de minimis' type approach was used for drinking water standards. Quite a landmark decision was that by the US EPA in the Drinking Water Act (US EPA, 1989c) when it was stated that drinking water should create no more than 1 extra gastro intestinal case per 10 000 people per year . Discussion which went into forming this decision was reported by Regli *et. al.* (1988). The basis of this limit was that the risk limit of 1 in 10 000 people per year was similar to that currently being experienced in the US and Canada through drinking water exposure, and was not out of proportion with other common microbial risks.

Earlier discussion of acceptable risks from microbials has pointed towards this kind of approach. In discussion of the risks of sludge disposal to land Akin *et. al.* (1977) concluded that it would be unrealistic to require all domestic wastes that are applied to land to be pathogen free. They highlighted the point that this could not be guaranteed without the complete testing of all waste with methods that were 100 percent efficient for all pathogens, which would be economically impractical and technically unattainable. The goal should therefore be to achieve and maintain the microbial hazard from waste disposal on land at an acceptable risk level. They concluded that for the health scientist, acceptable could be defined as when disease transmission through a single source could be demonstrated by epidemiology to occur below the background transmission rate of disease from all other sources.

In the absence of epidemiological information concerning the unrestricted marketing of biosolids, the use of a risk assessment approach is proposed. Risk assessment could be used to determine pathogen concentrations in biosolids which would raise the potential transmission rate above reported transmission rates.

Following on from this discussion the following principle for setting acceptable risks is proposed.

> **Principle 3.**
> **The risk of disease transmission through the re-use of biosolids products should be less than background transmission rates for that disease from other sources.**

In the case of biosolids re-use, the major limitation of this approach is that present biosolids re-use practices may contribute to background transmission rates of diseases. However, in the absence of epidemiological information it is not possible to know what this impact is.

It is also difficult to determine how much less than background transmission rates is acceptable. For the risk of mortality from individuals located near nuclear power plants it was considered that the risk should be 0.1% of the sum of prompt fatality risk resulting from other accidents to which members of the US population were generally exposed (Spangler, 1987). Similarly, it was considered that the risk of cancer fatalities to the population in the area near a nuclear power plant that might result from nuclear power plant operation should not exceed 0.1% of the sum of cancer fatality risks resulting from all other causes .

It is suggested that the rates of reported disease are a good baseline for setting criteria. However, 0.1% of risk from other sources as suggested by Spangler (1987) appears to be too conservative when applied to risk of disease, rather than risk of mortality. It is suggested that the criteria for biosolids should be based somewhere in the region of 1% to 10% of present infection rates, rather than 0.1%.

4.4.3 Exposure Assessment

Exposure pathways have been developed by the US EPA (1989b) for the land application, distribution and marketing of municipal sludge. These are shown below.

1. Sludge - Soil - Plant - Human Toxicity.
2. Sludge - Human Toxicity (Soil Ingestion).
3. Sludge - Soil - Plant - Animal - Human Toxicity.
4. Sludge - Animal (Direct Ingestion) - Human Toxicity.
5. Sludge - Soil - Plant - Animal Toxicity.
6. Sludge - Animal Toxicity (Direct Ingestion).
7. Sludge - Soil - Plant Toxicity.
8. Sludge - Soil -Soil Biota Toxicity.
9. Sludge - Soil - Soil Biota - Predator Toxicity.
10. Particulate Re suspension.
11. Surface Runoff.
12. Groundwater.

13. Vaporisation.

In the case of composted biosolids applied to the home garden, it is suggested that the most exposed individual would come from route 2, sludge - soil ingestion. It is also suggested that the most exposed and at risk individual would be a child ingesting biosolids while playing in the home garden. The number of children in the 1 to 3 years age group in Australia is approximately 520 000 (Castles, 1994). This at risk group therefore represents 3% of the total population of Australia. It was considered that adults handling biosolids were likely to ingest smaller amounts than children, and therefore be at less risk.

The exposure assessment was therefore based on soil ingestion by children, and is applied to each of the pathogens in Sections 4.5.1, 4.5.2 and 4.5.3 below.

The amount of soil which young children ingest has not been clearly established from published studies. Some early estimates were prepared by Lepow *et. al.* (1974) who measured the amount of soil on children's hands. By multiplying this figure by 10 they estimated the amount that would be ingested in one day as 100 mg. A far greater estimate was provided by Kimbrough *et. al.* (1984) with average daily soil ingestion value estimates ranging from 100 mg to 10 g per day depending on age (it was estimated that children aged 18 to 42 months ingested the most).

Soil ingestion studies have also been carried out using soil tracer methods. The earliest reported soil ingestion study for humans was conducted by Binder *et. al.* (1986). They used Al, Si and Ti as tracers in a study of 59 children aged 1 to 3 years. Their average daily soil ingestion estimates ranged from 181, 184 and 1834 for Al, Si and Ti respectively. The greatest limitation of this study was that food and other non soil sources were not measured.

Clausing *et. al.* (1987) measured soil ingestion rates in a group of 18 children aged 2 to 4, using the tracers Ti, Al and acid soluble residue (AIR). These were compared to a group of 6 children in hospital. The concentrations of tracers in faecal output from the hospital children was assumed to be from dietary and other non-soil sources. From this study the average daily soil ingestion was estimated to be 56 mg. In a later study by the same group of workers (van Wijnen *et. al.*, 1990) using the same tracers, but with 162 children, geometric mean soil ingestion values ranged from 0 to 200 mg/day, depending on age and location (some were in day care centres and some in a camping ground). In both these

studies the authors used a procedure of selecting the lowest soil ingestion value from the three tracers as their best estimate.

Calabrese *et. al.* (1989) conducted a study of 64 children ranging in age from 1 to 4 years using 8 tracers. They reported that the median values from the three most reliable tracers were 29, 40 and 9 mg/day from Al, Si and Y. Median values were reported because mean values were skewed by one child who ingested between 5 and 8g/day.

Davis *et. al.* (1990) used Al, Si and Ti as tracer elements in a study of 104 children aged between 2 and 7 years. Food consumption was measured for each child. Average daily soil ingestion values were 39 mg, 82 and 245 for Al, Si and Ti respectively.

Calabrese and Stanek (1991) evaluated the above studies on the basis of their potential accuracy and precision and suggested that the above results are unreliable. However, their analysis was based on an adult study of only 6 replicates (Stanek and Calabrese, 1991).

The data produced on soil ingestion by children does not lend itself to statistical analysis because of the widely different procedures used. However, from the studies above a semi quantitative average estimate of daily soil ingestion by children of 50 mg was estimated.

However, as reported by Chaney (1993), within children there are a group called pica who have abnormal mouthing behaviour. Of the sixty five children studied by Calabrese *et al.* (1989) one had pica behaviour and ingested somewhere in the region of 5 to 8 g of soil per day. In the study of van Wijnen *et. al.* (1990) 9 out of 557 daily soil ingestion values were greater than 1 g/day. In the study of Clausing *et. al.* (1987) none of the 18 children had soil ingestion values that were exceptionally high for all three tracers. The other studies did not provide enough information to identify pica children. From the three studies for which data was available it was calculated that approximately 1 in 60 or 1.7% of children ingested abnormally high levels of soil. Abnormally high ingestion rates were considered to be greater than 1 g per day.

On the basis of these studies an average 'best guess' estimate for soil ingestion was taken to be 50 mg soil/day. For 1.7% of children soil ingestion may be as high

as 8 g/day. It was estimated that an average pica child would ingest 5 g of soil/day.

4.5 CRITERIA FOR *SALMONELLA*

4.5.1 Acceptable Limit for *Salmonella* Infection From Composted Biosolids

Setting acceptable limits for *Salmonella* in composted biosolids is made difficult by the possibility that low levels of salmonellae may regrow during the storage of composted biosolids or biosolids products.

As suggested by Russ and Yanko (1981) it may be possible to predict the *Salmonella* regrowth potential of biosolids products as a function of the carbon/nitrogen ratio and moisture level. Jiminez and Garcia (1991) also recommended that the carbon/nitrogen ratio in combination with cation exchange capacity could be used as a measure of biological stability. This is an area that needs further investigation but in the absence of further information it does not appear feasible to incorporate the effect of regrowth potential on criteria setting.

As an alternative approach it is suggested that sensible precautions should be taken to try and prevent regrowth. A maturation period should be specified following composting. Further work needs to be carried out to determine the required length for a maturation period. Products should be monitored for salmonellae before bulk removal for sale or before bagging. A random monitoring programme should be conducted for bagged products.

For devising acceptable limits for salmonellae in composted biosolids products it was decided to make the assumption that background rates of transmission for *Salmonella* are acceptable to the public. What impact present biosolids disposal practices have on reported cases has not been assessed, but as discussed in Section 4.4.2, these may contribute to present background transmission rates.

In 1991 the annual reported *Salmonella* infection rate for children in Australia in the 0 to 4 age group was approximately 400 per 100 000 (Anura and Hall, 1992). This means that the probability of infection was 4×10^{-3}. The average notification rate for the whole population was 31 in 100 000 with a probability of infection of 3×10^{-4}.

As described in Section 4.4.2, it was decided that risks of infection from *Salmonella* in biosolids should be at most in the region of 1% to 10% of reported infection rates. Therefore the probability of a child becoming infected with *Salmonella* from ingesting biosolids in the home garden should be at most in the region of 4×10^{-4} to 4×10^{-5}.

4.5.2 Infectious Dose for *Salmonella*

The number of organisms which need to be ingested to cause an infection (infectious dose) is not a constant number. It varies with a number of factors including:

1. The species of *Salmonella.*
2. The status of the individual. Infants and the elderly appear to be more susceptible to infection as do those who already have another infection. Other factors include the possibility of immunity and immune suppressing effects of some medication, such as antacids.
3. The vehicle in which *Salmonella* is administered. It appears that fatty foods such as chocolate and cheese protect *Salmonella* from acid attack in the stomach and enable survival to the intestinal tract.

For this reason it is not possible to say what the infectious dose for *Salmonella* will be in individual cases.

The range within which the infectious dose for *Salmonella* is likely to fall can be derived from two sources. These are human volunteer studies and outbreaks. Both types of studies suggest that higher doses of *Salmonella* appear to cause a higher rate of attack and quicker onset of infection. However, there appears to be some discrepancies between results from the two types of studies. As summarised by Blaser and Newman (1982) the lowest dose of ingested *Salmonella* reported to cause infection in human volunteer studies was 10^5 *Salmonella* per mL, which resulted in attack rates ranging from 17 to 35%. When reference to infectious dose is made it is usually based on data from human volunteer studies.

Haas (1983) used infectious dose data reported by Hornick *et al.* (1970) to test whether dose-response models could be fit to data from a human volunteer study. The conclusion was that the beta distributed effectiveness model could be

used to describe the available data for *Salmonella*. The data initially used by Haas (1983) to fit the models is shown in Table 2.

Table 2. Data used by Haas (1983) for Testing *Salmonella* Infectious Dose Model

Organism and Number of Organisms Ingested	Response	
	Positive	Negative
Salmonella typhosa		
10^3	0	14
10^5	32	84
10^7	16	16
10^9	40	2

Infectious dose information from outbreaks, as opposed to human dosing studies, appears to provide a different picture and this is shown in Table 3.

Table 3 shows that in the outbreaks reported by Blaser and Newman (1982) it appeared that *Salmonella* infections were caused by materials contaminated with as low as 17 organisms. In more than half of the 13 outbreaks recorded the infectious dose appeared to be less than 1000 organisms. In a more recent study reported by D'Aoust (1985) the infectious dose from six cases caused by contaminated cheese appeared to be less than 10. In the case of an elderly woman infection appeared to be caused by ingesting 1 *Salmonella*. In the reported outbreaks it is possible that the calculated concentration of *Salmonella* in the infectious material was underestimated. However, there is no direct evidence for this.

Table 3. Calculated Doses of *Salmonella* in a Number of Outbreaks (taken from Blaser and Newman, 1982 and *D'Aoust, 1985).

Vehicle	No. of Patients	Estimated No. of Organisms Ingested
Water	16 000	17
Pancreatin	1	44
Pancreatin	1	200
Oral Vaccine	7 to 10	60 to 90
Hamburger	46	60 to 230
Chocolate Balls	95	100
Chocolate Balls	114	250
Cheddar Cheese	339	100 to 500
Imitation Ice Cream	1 790	11 000
Carmine Dye Capsules	28	15 000 to 60 000
Goat Cheese	6	150 000
Ham	8	1×10^6 to 2×10^6
Goat Cheese	5	1×10^{11}
*Cheddar Cheese	> 1 500	0.7 to 6.1×10^0

Ideally a probability distribution showing the probability of infection from a range of concentrations should be created using the data from outbreaks, as has been shown by Haas (1983) for data from human volunteer studies. However, the data shown in Table 3 above are not sufficient to do this. The data do not show the rate of infection per exposure for different exposure levels. However, a very crude probability distribution has been produced by listing the number of outbreaks reported to be caused by a range of *Salmonella* concentrations, as shown in Table 4. This table also shows the percentage of the total number of 14 outbreaks which reported infection at that dose.

Table 4. Number of Outbreaks Caused by a Range of *Salmonella* Concentrations (Data taken from Blaser and Newman, 1982 and D'Aoust, 1985)

Infectious Dose Less Than:	Number	Percentage
1	1	7
10	1	7
10^2	5	36
10^3	9	64
10^4	9	64
10^5	11	79
10^6	12	86
10^7	13	93
10^{11}	14	100

Table 4 shows that ingestion of 1 salmonellae resulted in disease in 1 of the 14 reported outbreaks and ingestion of 100 salmonellae resulted in disease in 5 out of the 14 outbreaks. The data is not sufficient to be used for calculating probability distributions but shows that the data used by Haas (1983) could result in underestimation of the probability of infection.

As the outbreak data is not sufficient to generate a probability distribution model the beta distributed probability model generated by Rose and Gerba (1991) from human volunteer data appears to be the best available method of calculating probability of infection based on dose. For *Salmonella* the model is

$$p = 1 - (1+(N/139.9))^{-0.33} \qquad (1)$$

where
p = probability of infection and
N = exposure

4.5.3 Risk Characterisation

Equation 1 (above) was expressed in terms of N so that acceptable exposure levels could be calculated from acceptable probabilities, as shown below.

$$N = 139.9((1-p)^{-3.03} - 1) \qquad (2)$$

The acceptable exposure was then calculated for the range of acceptable probabilities of infection derived in Section 4.4.2. This was then expressed as a limit for *Salmonella* in biosolids material, for a normal child and a pica child, as shown in Table 5 below.

Table 5. Acceptable Limits for *Salmonella* in Composted Biosolids

Acceptable Probability of Infection	Acceptable Exposure (No. of Organisms)	Amount Ingested	Limits for *Salmonella* in Composted Biosolids
4×10^{-4}	0.169	0.05 g (normal child)	1 in 0.3 g
4×10^{-4}	0.169	5 g (pica child)	1 in 30 g
4×10^{-5}	0.0169	0.05 g	1 in 3 g
4×10^{-5}	0.0169	5 g	1 in 300 g

4.5.4 Risk Criteria

Table 5 shows that the acceptable limit for *Salmonella* in biosolids products ranges from 1 in 0.3 g to 1 in 300 g depending on which parameters are chosen.

For determining which of these criteria would be acceptable the first principle suggested is that the pica child should be protected. Although the number of exposed individuals in this group may be low, it is felt that these most at risk individuals should be protected.

It is difficult to say what an acceptable probability of infection is. However, it is proposed that a criteria of less than 1 *Salmonella* detected in 50 g of biosolids products is acceptable. This is partially an acknowledgement of the limitations of methodology, and financial limitations on analysing larger quantities of biosolids material. The limit of less than 1 *Salmonella* in 50 g of biosolids would result in

a probability of infection of less than 2×10^{-4} for a pica child, which is less than 6% of present reported infection rates for children in the 0 to 5 age group.

This suggested limit of less than 1 *Salmonella* detected in 50 g of biosolids product differs from that in US EPA sludge guidelines (US EPA, 1992). US EPA guidelines specify that sewage sludge which is sold or given away must contain less that 3 *Salmonella* sp. per 4 grams total solids sewage sludge (US EPA, 1992).

These two limits differ in a number of ways. Firstly US guidelines specify a limit for *Salmonella* per total solids, which refers to dry weight. The suggested limit in this paper is per gram of biosolids product as produced, or wet weight. The reason for setting a limit per gram of wet weight is that it is conceptually easier to apply and less analysis is required than for dry weight. US guidelines have specified limits per gram dry weight in the past because there can be an apparent increase in concentrations through treatment such as dewatering if wet weights are used. However, for routine monitoring of one type of product (composted biosolids) this is not an important issue.

Secondly the two limits differ in the actual acceptable concentrations specified. If composted biosolids contained approximately 50% solids, then the US limit would be equivalent to less than 1 *Salmonella* per 2.7 g of composted biosolids. In a mixed product containing 25% composted biosolids this would be a limit of approximately less than 1 *Salmonella* per 10 g of biosolids product. The US guidelines are therefore less stringent than suggested in this risk assessment paper, as this would equate to a limit of less than 5 *Salmonella* per 50 g of biosolids product. The reason for the difference is that US guidelines are based on detectable limits whereas this paper uses a risk assessment approach. However, lower concentrations of *Salmonella* can be detected than the suggested detectable limit specified in US guidelines. The limit of less than 1 *Salmonella* in 50 g of product which is suggested on the basis of risk assessment is methodologically achievable.

The third difference is that US guidelines specify a MPN or quantitative method where as this paper recommends a presence/absence test. In both US guidelines and this paper it is suggested that *Salmonella* concentrations should be below a certain concentration. This can be shown by a presence/absence test. A presence/absence test will be more cost effective than an MPN test.

A suggested method for carrying out a presence/absence test for *Salmonella* in 50 g of composted biosolids product is that described by Hu, Gibbs and Ho (1995).

4.6 CRITERIA FOR *GIARDIA*

4.6.1 Acceptable limit for *Giardia* Infection From Composted Biosolids

At present there is no published information on the effect of composting on *Giardia* concentrations in biosolids. Extremely high numbers of *Giardia* cysts have been detected in anaerobically digested biosolids (Gibbs *et. al.*, 1995). Unpublished data suggests that cysts are not removed by the composting process (Mort, personal communication).

However, the important unanswered question with regard to *Giardia* is what effect composting has on the viability or infectivity of cysts. Techniques used in the few studies which have investigated *Giardia* concentrations in biosolids have not been adequate to assess viability.

The approach which has been taken in US Drinking Water Guidelines (US EPA, 1989c) is to consider that cysts detected using microscopy are potentially viable. Studies have not been conducted to determine whether this is a reasonable assumption.

Two studies on the effect of anaerobic digestion on *Giardia* cysts have suggested that cyst viability is greatly reduced by anaerobic digestion (Gavaghan *et. al.*, 1993, van Praagh *et. al.*, 1993). These studies were conducted using laboratory scale digesters with seeded *Giardia muris* (mouse) isolates. How representative these are of full scale processes with indigenous human isolates is uncertain.

Reported infection rates in the community are high. Data is not available for the most at risk sub population group, young children, but reported infection rates for the whole community were 66 per 100 000 in 1991 in Western Australia (Public Health and Diseases Unit of the State Health Laboratory Services, 1992). The probability of infection from all sources was therefore 7×10^{-4}. It is likely that the reported prevalence in the 1 to 5 age group is higher than this. In a study of giardiasis in Mount Isa, Queensland, Boreham and Phillips (1986) found that 4.6% of the population were infected with *Giardia*, with 12% of children in the 1 to 5 years age group infected. Symptoms of giardiasis also appeared to be more

prevalent in children than in the whole population. Similarly in a study of the prevalence of *Giardia* in Aboriginal communities, 32% of children and 12.5% of adults were infected with *Giardia* (Meloni *et. al.*, 1993).

If the suggested risk of infection from *Giardia* from biosolids use is to present a risk less than from 1 to 10% of background reported infection rates, as discussed in Section 4.4.2, then the probability of risk should be less than between 7.0×10^{-5} to 7.0×10^{-6}.

4.6.2 Infectious Dose for *Giardia*

Very few studies have been conducted to determine the infectious dose for *Giardia*. However, one of the few human dosing studies which has been conducted suggests that the ingestion of one *Giardia* cyst may be enough to cause infection (Rendtorff, 1954).

An analysis of dose-response data by Rose and Gerba (1991) resulted in the development of a dose-response probability model as described previously for *Salmonella* (Sections 4.5.2 and 4.5.3).

The two equations for *Giardia* are shown below.

Single-hit exponential model

$$p = 1 - \exp(-0.0199N) \qquad (3)$$

$$N = -\ln(1-p)/0.0199 \qquad (4)$$

where
p = probability of infection and
N = exposure

4.6.3 Risk Characterisation

Using the acceptable probabilities of infection (shown in Section 4.6.1), of between 1% and 10% of reported infection rates, and acceptable exposure levels, acceptable limits for *Giardia* in composted biosolids were calculated for normal and pica children as shown in Table 6 below.

Table 6. Acceptable Limits for *Giardia* in Composted Biosolids

Acceptable Probability of Infection	Acceptable Exposure (No. of Organisms)	Amount Ingested	Limits for *Giardia* in Composted Biosolids
7×10^{-5}	3.5×10^{-3}	0.05 g (normal child)	1 in 14 g
7×10^{-5}	3.5×10^{-3}	5 g (pica child)	1 in 1400 g
7×10^{-6}	3.5×10^{-4}	0.05 g	1 in 140 g
7×10^{-6}	3.5×10^{-4}	5 g	1 in 14 000 g

4.6.4 Risk Criteria

If the assumption is made that every cyst detected in biosolids products is viable, then depending on the assumptions made, the acceptable limits range between 1 in 14 g to 1 in 14 000 g, as shown in Table 6.

Based on results from monitoring studies carried out thus far, the use of any of the criteria shown above would preclude the re-use of biosolids which had been treated by composting. The average concentration of *Giardia* in digested, dewatered biosolids was 500/g (Gibbs *et. al.*, 1995) and in composted biosolids data suggest that numbers are high following composting (Gibbs, unpublished data).

As the only study which has assessed viability suggested that *Giardia* cysts may be rendered non viable by anaerobic digestion, it does not seem prudent at this stage to recommend that any of the above criteria should be included in biosolids guidelines. However, it is highly recommended that further research should be carried out to evaluate the effect of composting on *Giardia* cyst viability.

4.7 ENTERIC VIRUSES

At this point in time it is felt that reasonable criteria for acceptable concentrations of enteric viruses in biosolids products could not be set. Methods for monitoring

viable enteric virus concentrations in biosolids are not developed enough to justify any method involving routine monitoring of biosolids for viruses.

It is suggested that more work should be carried out on methods for detecting enteric viruses in biosolids, and research carried out on the effect of composting on virus concentrations in biosolids.

5. CONCLUSIONS

1. Epidemiological studies, laboratory studies or field studies have not provided evidence that biosolids composted according to US EPA recommendations will be free of pathogens.

2. A risk assessment approach should therefore be used to develop criteria for acceptable concentrations of pathogens in biosolids products.

3. The application of risk assessment techniques to developing pathogen standards for biosolids re-use is difficult, because principles for risk assessment are not well developed for microbials, and data is limited.

4. A suggested principle is that the risk of disease transmission through the re-use of biosolids products should be less than background transmission rates for that disease from other sources.

5. Risks should be calculated for the most at risk sub population, which was suggested to be children accidentally ingesting biosolids in the home garden.

6. Composted biosolids products should be monitored for the presence of *Salmonella*, but not for *Giardia* or enteric viruses. Methods for *Giardia* and enteric viruses are not sufficiently developed to justify the inclusion of monitoring requirements for these pathogens.

6. RECOMMENDATIONS

6.1 BIOSOLIDS GUIDELINES

1. Guidelines for biosolids management should include a requirement for monitoring biosolids products for the presence of *Salmonella,* as the composting process does not appear to ensure the complete removal of *Salmonella.* It is recommended that the limit for *Salmonella* in biosolids product should be less than 1 *Salmonella* detected in 50 g of biosolids product. A suggested method is that described by Hu, Gibbs and Ho (1995).

2. As *Salmonella* regrowth may occur in biosolids products, guidelines should include requirements for a maturation period following composting to allow product stabilisation. Final products should be monitored before bulk removal or bagging. A random monitoring programme should be conducted for bagged products.

3. A requirement for routine monitoring biosolids products for *Giardia* or enteric viruses should not be included in guidelines until methods are further developed.

6.2 FURTHER RESEARCH

The following recommendations for further research are listed in order of priority.

1. An epidemiological study of the unrestricted marketing of biosolids products should be carried out. This would provide stronger evidence of the risks associated with the unrestricted marketing of composted biosolids products than the risk assessment approach described here. Valuable information could be obtained by carrying out a prospective study in which purchasers of biosolids products were surveyed for reported illness and compared to a control group.

2. It has been demonstrated that *Salmonella* regrowth can occur in composted biosolids products. Processes to control this regrowth

need to be investigated. This area of research is currently being funded by WAWA and carried out at Murdoch University with the project 'Regrowth of Pathogens in Composted Sludge'.

3. Future research should be conducted to develop methods for determining *Giardia* viability in biosolids products, and these methods should be applied to studying the effect of composting on *Giardia* viability. This research is essential to gain a better understanding of the risks associated with *Giardia* as the assumption that all *Giardia* cysts are viable would prohibit the re-use of composted biosolids. This research is presently being funded by WAWA and carried out at Murdoch University with the project 'Presence and Viability of *Giardia* in Composted Sludge'.

4. It appears likely that actual virus concentrations in biosolids are higher than can presently be measured. Research should be conducted to improve methods for detecting concentrations of infectious virus particles in biosolids products, and then applied to studying the effect of composting on enteric viruses. As a preliminary step a project could be developed to compare PCR methods to culture methods for enteric viruses.

5. At present there is uncertainty about the level of pathogen reduction which can be achieved by a fully operational windrow composting system. The efficiency of windrow composting for pathogen removal should be fully investigated using a full-scale operational process. A survey of biosolids composting facilities from different sites in Australia has been proposed to WAWA.

6. It has been suggested in this risk assessment paper that a compost maturation period should be specified in biosolids guidelines. This is to allow product stabilisation to prevent regrowth. Research should be carried out to determine whether this is an effective strategy and to determine the optimum length of maturation. This research could be a monitoring programme of stored composted products carried out in conjunction with an operator.

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